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	L8	(L7 and in vitro)	48		
	L7	L4 and (drug selection)	54		
Γ	L6	(L4 and (drug selection) or (drug screening))	19107		
	L5	L4 and (drug selection) or (drug screening)	19107		
Γ	L4	(androgen receptor mutation) and (anti-androgen) or (antiandrogen)	3631		
Γ	L3	(L2 and antiandrogen selection)	. 0		
Γ	L2	L1 and mutation and (drug selection)	40		
Γ	L1	(androgen receptor) and (anti-androgen) or (antiandrogen)	3881		

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<u>#52</u>	Search (androgen antagonist) and (screening) and (cell proliferation) and (in vitro) Limits: Publication Date to 2002/6/3	17:55:01	<u>20</u>
#48	Search (androgen antagonist) and (screening) and (cell proliferation) Limits: Publication Date to 2002/6/3	17:54:43	46
<u>#47</u>	Search (androgen antagonist) and (screening) Limits: Publication Date to 2002/6/3	17:44:05	<u>138</u>
<u>#46</u>	Search joly-pharaboz and r2 Limits: Publication Date to 2002/6/3	13:41:57	2
<u>#45</u>	Search joly-pharaboz Limits: Publication Date to 2002/6/3	13:41:41	13
. #43	Search (Androgen Receptor) and (selection) and mutation and antiandrogen Limits: Publication Date to 2002/6/3	13:03:41	2
#41	Search (Androgen Receptor) and (selection) and mutation Limits: Publication Date to 2002/6/3	12:52:16	<u>17</u>
<u>#40</u>	Search (Androgen Receptor) and (selection) and mutation	12:51:43	<u>27</u>
<u>#36</u>	Search AR and (drug selection) and mutation	12:48:42	<u>12</u>
<u>#35</u>	Search AR and (drug selection)	12:48:27	<u> 187</u>
#34	Search (AR and (drug selection)	12:48:21	***********
<u>#23</u>	Search (AR gene mutation) or (AR mutation) and drug selection	12:39:48	12
<u>#20</u>	Search bentel and ar	12:29:02	11
<u>#19</u>	Search bentel	12:28:52	
<u>#14</u>	Search antiandrogen and (androgen receptor mutation) Limits: Publication Date to 2002/6/3	11:45:59	60
<u>#17</u>	Search antiandrogen and (androgen receptor mutation) and culture Limits: Publication Date to 2002/6/3	11:34:36	1

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#16 Search LNCaP Limits: Publication Date to 2002/6/3	11:34:14	<u>1637</u>
#13 Search antiandrogen and androgen receptor mutation Limits: Publication Date to 2002/6/3	n 11:29:03	60
#11 Search AWS and mutation Limits: Publication Date t 2002/6/3	o 11:28:06	<u>3</u>
#10 Search AWS and antiandrogen and mutation Limits: Publication Date to 2002/6/3	11:26:16	<u>0</u>
#9 Search AWS and antiandrogen and androgen receptor mutation Limits: Publication Date to 2002/6/3	11:26:03	0
#5 Search antiandrogen drug and androgen receptor mutation and resistance Limits: Publication Date to 2002/6/3	11:21:21	7
#3 Search antiandrogen drug and androgen receptor mutation Limits: Publication Date to 2002/6/3	11:18:38	<u>34</u>
#2 Search antiandrogen drug and androgen receptor mutation	11:18:15	<u>67</u>



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B 155, 159, 10, 203, 35, 5, 467, 73, 434, 34
       04may07 15:29:55 User290558 Session D105.1
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          (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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11309067
          PMID: 9111707
Androgen receptor gene mutations in prostate cancer. Implications for
disease progression and therapy.
  Culig Z; Hobisch A; Hittmair A; Cronauer M V; Radmayr C; Bartsch G;
 Department of Urology, University of Innsbruck, Austria.
                                Jan 1997, 10 (1) p50-8, ISSN 1170-229X
 Drugs & aging (NEW ZEALAND)
        Journal Code: 9102074
  Publishing Model Print
 Document type: Journal Article; Review
 Languages: ENGLISH
 Main Citation Owner: NLM
 Record type: MEDLINE; Completed
 Subfile:
           INDEX MEDICUS
 Recent studies indicate that androgen receptors are present in all
histological types of prostatic tumours, in relapsed prostatic carcinomas
and in tumour metastases, even those obtained from patients in whom
endocrine therapy was unsuccessful. Several research groups have asked
whether structurally altered androgen receptors might be present in human
prostatic tumours. The first androgen receptor mutation in prostate cancer
was detected in the tumour cell line LNCaP. The frequency of androgen
receptor mutations in primary tumours of the prostate is relatively low. In
contrast, a high frequency of mutations has been reported in bone
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metastases from patients who did not respond to endocrine therapy. This fact may reflect genetic instability in these late tumour stages. Mutant androgen receptors detected in human prostate cancer cells are 'promiscuous receptors'; that is, they are activated not only by synthetic and testicular androgens, but also by adrenal androgens, products of dihydrotestosterone metabolism, estrogenic and progestagenic steroids, and by nonsteroidal antiandrogens. Interestingly, the nonsteroidal antiandrogens hydroxyflutamide and nilutamide, but not bicalutamide, have been reported to have agonistic effects at mutant androgen receptors. It is speculated that the existence of androgen receptor mutations may explain, at least in part, the 'antiandrogen withdrawal syndrome': a temporary improvement in a subpopulation of prostate cancer patients following cessation of an antiandrogen from a therapeutic protocol. Further studies on androgen receptor alterations in prostate cancer should focus on metastatic specimens obtained from the late stages of this disease. (69 Refs.)

Tags: Male

Descriptors: *Mutation; *Prostatic Neoplasms--genetics--GE; *Receptors, Androgen--genetics--GE; Humans; Prostatic Neoplasms--therapy--TH; Receptors, Androgen--analysis--AN; Research Support, Non-U.S. Gov't; Structure-Activity Relationship; Tumor Cells, Cultured

CAS Registry No.: 0 (Receptors, Androgen)

Record Date Created: 19970508
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16983697 BIOSIS NO.: 200200577208

Abolition of hypertension-induced end-organ damage by androgen receptor blockade in transgenic rats harboring the mouse Ren-2 gene

AUTHOR: Baltatu Ovidiu; Cayla Cecile; Iliescu Radu; Andreev Dmitrii; Jordan Cynthia; Bader Michael (Reprint)

AUTHOR ADDRESS: Max-Delbrueck-Center for Molecular Medicine (MDC),

Robert-Roessle-Str. 10, Berlin-Buch, D-13092, Germany**Germany

JOURNAL: Journal of the American Society of Nephrology 13 (11): p2681-2687 November, 2002 2002

MEDIUM: print ISSN: 1046-6673

DOCUMENT TYPE: Article RECORD TYPE: Abstract LANGUAGE: English

ABSTRACT: A sexual dimorphism in hypertension has been observed in both human and laboratory animal studies. The mechanisms by which male sex hormones regulate cardiovascular homeostasis are still not yet fully understood and represent the subject of this study. The possible involvement of androgen receptors in the development of hypertension and end-organ damage in transgenic rats harboring the mouse Ren-2 renin gene (TGR(mREN2)27) was studied. Male TGR(mREN2)27 rats were treated with the androgen receptor antagonist Flutamide starting at 4 wk of age. Also, an androgen receptor mutation (testicular feminization mutation (tfm)) was introduced in these rats by crossbreeding male TGR(mREN2)27 rats with tfm rats. The resulting offspring male rats that contain the tfm mutation are insensitive to androgens. Flutamide treatment or tfm mutation produced a significant attenuation of the development of hypertension. Besides a reduction in cardiac hypertrophy, urinary albumin excretion was blunted and no histologic characteristics of end-organ damage were observed in

15-fold after Flutamide treatment and 2.7-fold by the tfm mutation. Also, plasma estrogens and luteinizing and follicle-stimulating hormones were significantly increased. Plasma renin concentrations and activity but not plasma angiotensinogen were reduced. Our results indicate that androgens contribute not only to the development of hypertension, but even more importantly to end-organ damage in TGR(mREN2)27 rats. REGISTRY NUMBERS: 9002-68-0: FSH; 13311-84-7: flutamide; 9015-94-5: renin; 58-22-0: testosterone DESCRIPTORS: MAJOR CONCEPTS: Cardiovascular System -- Transport and Circulation; Endocrine System -- Chemical Coordination and Homeostasis; Molecular Genetics -- Biochemistry and Molecular Biophysics; Pharmacology BIOSYSTEMATIC NAMES: Muridae--Rodentia, Mammalia, Vertebrata, Chordata, Animalia ORGANISMS: mouse (Muridae); rat (Muridae)--transgenic ORGANISMS: PARTS ETC: kidney--excretory system COMMON TAXONOMIC TERMS: Animals; Chordates; Mammals; Nonhuman Vertebrates ; Nonhuman Mammals; Rodents; Vertebrates DISEASES: end-organ damage--disease-miscellaneous; hypertension--vascular disease MESH TERMS: Hypertension (MeSH) CHEMICALS & BIOCHEMICALS: FSH--hormone; LH {luteinizing hormone}-hormone; androgen receptor; estrogen--hormone; flutamide-antiandrogen-drug, hormone-drug; renin; testosterone--androgen GENE NAME: mouse Ren-2 gene (Muridae) MISCELLANEOUS TERMS: sexual dimorphism; testicular feminization mutation CONCEPT CODES: 03502 Genetics - General 03506 Genetics - Animal 10064 Biochemistry studies - Proteins, peptides and amino acids 10067 Biochemistry studies - Sterols and steroids 10802 Enzymes - General and comparative studies: coenzymes 12512 Pathology - Therapy 14504 Cardiovascular system - Physiology and biochemistry 14508 Cardiovascular system - Blood vessel pathology 15504 Urinary system - Physiology and biochemistry 17002 Endocrine - General 17014 Endocrine - Pituitary 22002 Pharmacology - General 22016 Pharmacology - Endocrine 35500 Allergy **BIOSYSTEMATIC CODES:** 86375 Muridae (Item 1 from file: 34) DIALOG(R)File 34:SciSearch(R) Cited Ref Sci (c) 2007 The Thomson Corp. All rts. reserv. Number of References: 32 Genuine Article#: XW121 Title: Similar clinical outcomes in African-American and non-African-American males treated with suramin for metastatic prostate

Author(s): Bergan RC (REPRINT); Walls RG; Figg WD; Dawson NA; Headlee D;

Corporate Source: NCI, DEPT CELL & CANC BIOL, BLDG 10, ROOM

the kidney after Flutamide treatment. Testosterone levels increased

Tompkins A; Steinberg SM; Reed E

12N226/BETHESDA//MD/20892 (REPRINT)

Journal: JOURNAL OF THE NATIONAL MEDICAL ASSOCIATION, 1997, V89, N9 (SEP), P622-628

ISSN: 0027-9684 Publication date: 19970900

Publisher: SLACK INC, 6900 GROVE RD, THOROFARE, NJ 08086

Language: English Document Type: ARTICLE

Geographic Location: USA

Subfile: CC CLIN--Current Contents, Clinical Medicine; Journal Subject Category: MEDICINE, GENERAL & INTERNAL

Abstract: African-American males have a higher incidence of prostate cancer than non-African-American males and an overall poorer prognosis. Environmental factors such as socioeconomic status and biological factors such as an increased frequency of androgen receptor mutation have been identified as causal. As androgen ablation therapy is ubiquitous in the treatment of metastatic prostate cancer, little information is available on clinical outcome independent of hormone therapy. Our experience at the Warren G. Magnusson Clinical Center, National Institutes of Health with the anticancer agent, suramin, offers the opportunity to study clinical outcome in patients treated with an agent whose tumoricidal activity is not dependent on androgen receptor function.

Clinical outcome was examined retrospectively in 43 patients treated on a single suramin-based protocol and evaluated as a Function of ethnic background. No significant difference in time to disease progression or survival was observed between African Americans (n=4) and the other 39 patients. These findings are consistent with the hypothesis that therapies that work through mechanisms independent of the androgen receptor may result in similar outcomes across ethnic groups.

Descriptors--Author Keywords: prostate cancer; suramin; African Americans; ethnic groups; clinical outcome

Identifiers--KeyWord Plus(R): ANDROGEN RECEPTOR GENE; FLUTAMIDE WITHDRAWAL; WHITE MEN; GROWTH; BLACK; PROLIFERATION; ANGIOGENESIS; INHIBITION; CARCINOMA; MUTATION

Research Fronts: 95-0444 002 (METASTATIC PROSTATE-CANCER; SURAMIN THERAPY; ANTIANDROGEN WITHDRAWAL SYNDROME)

95-4637 002 (PROGNOSTIC FACTORS; CHILDRENS CANCER GROUP RANDOMIZED TRIAL; PROLONGED SURVIVAL; SUPRATENTORIAL PRIMITIVE NEUROECTODERMAL TUMORS)

95-0854 001 (SHORT TANDEM REPEAT LOCI; DNA TYPING; D1S80 POPULATION-DATA; STR MULTIPLEX SYSTEM; MODERN HUMAN ORIGINS) Cited References:

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PARKER SL, 1996, V46, P5, CA CANC J CLIN
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SARTOR O, 1994, V86, P222, J NATL CANCER I
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SCHRELL UMH, 1995, V82, P600, J NEUROSURG
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VELDSCHOLTE J, 1990, V173, P534, BIOCHEM BIOPH RES CO
VIJAYAKUMAR S, 1992, V1, P541, CANCER EPIDEM BIOMAR
WADE TP, 1992, V53, P195, J SURG RES

2/9/4 (Item 2 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
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05179738 Genuine Article#: VF423 Number of References: 79 Title: THE ANDROGEN RECEPTOR IN PROSTATE-CANCER

Author(s): TRAPMAN J; BRINKMANN AO

Corporate Source: ERASMUS UNIV ROTTERDAM, DEPT PATHOL, POB 1738/NL-3000 DR ROTTERDAM/NETHERLANDS/; ERASMUS UNIV ROTTERDAM, DEPT ENDOCRINOL & REPROD/NL-3000 DR ROTTERDAM/NETHERLANDS/

Journal: PATHOLOGY RESEARCH AND PRACTICE, 1996, V192, N7 (JUL), P752-760

ISSN: 0344-0338

Language: ENGLISH Document Type: ARTICLE

Geographic Location: NETHERLANDS

Subfile: SciSearch; CC LIFE--Current Contents, Life Sciences

Journal Subject Category: PATHOLOGY

Abstract: The androgen receptor is a member of the family of nuclear receptors. In its activated form as an androgen receptor ligand complex (the ligand can either be testosterone or 5 alpha-dihydrotestosterone), the androgen receptor is able to regulate a specific expression of target genes. The androgen receptor is expressed at high levels in male reproductive tissues. Mutations in the androgen receptor gene are the molecular cause of the androgen insensitivity syndrome, which is characterized by an abcrrant male or an apparently female phenotype. Expansion of a CAG-repeat, encoding a polymorphic glutamine stretch is the cause of a rare motor neuron disease (Kennedy's disease).

Hormonal therapy is the treatment of choice for metastatic prostate cancer. Hormone refractory prostate tumors in general still express androgen receptor in a proportion of the late stage prostrate tumors, somatic mutations in the androgen receptor gene have been described. Mutations can result in diminished ligand specificity of the androgen receptor. Furthermore, it has been hypothesized that ligand independent mechanisms can also be involved in androgen receptor activation.

Descriptors--Author Keywords: PROSTATE CANCER; ANDROGEN RECEPTOR; MUTATION; STRUCTURE; FUNCTION

Identifiers--KeyWords Plus: KERATINOCYTE GROWTH-FACTOR; STEROID-HORMONE
 RECEPTORS; GENE-MUTATIONS; DNA-BINDING; GLUCOCORTICOID RECEPTOR;
 EPITHELIAL INTERACTIONS; FLUTAMIDE WITHDRAWAL; RESPONSE ELEMENT;
 LIGAND-BINDING; IMAGE-ANALYSIS

Research Fronts: 94-0655 003 (RETINOIC ACID RECEPTORS; RESPONSE ELEMENT SELECTIVITY; ISOFORM-SPECIFIC AMINO-TERMINAL DOMAINS DICTATE DNA-BINDING PROPERTIES OF ROR-ALPHA)

94-0980 001 (TRINUCLEOTIDE REPEAT EXPANSION IN NEUROLOGICAL DISEASE;

GENE LOCATION; MYOTONIC-DYSTROPHY MUTATION; NEUROMUSCULAR DISORDERS; FRAGILE-X LOCUS) 94-1169 001 (ANDROGEN RECEPTOR GENE; ALTERED C-MYC EXPRESSION IN PROSTATE-CANCER CELLS; NONISOTOPIC SINGLE-STRAND CONFORMATION POLYMORPHISM ANALYSIS) (METASTATIC PROSTATE-CANCER; ENDOCRINE COMBINATION THERAPY; TOTAL ANDROGEN BLOCKADE; NONSTEROIDAL ANTIANDROGEN NILUTAMIDE) Cited References: ADLER AJ, 1991, V5, P1587, MOL ENDOCRINOL ALARID ET, 1994, V91, P1074, P NATL ACAD SCI USA BEATO M, 1995, V83, P851, CELL BENTVELSEN FM, 1995, V113, P245, MOL CELL ENDOCRINOL BORING CC, 1994, V44, P7, CA-CANCER J CLIN BRINKMANN AO, 1992, V14, P95, CANCER SURV CHODAK GW, 1992, V147, P798, J UROLOGY CHUNG LWK, 1991, V11, P91, CANCER SURV CLAESSENS F, 1989, V164, P833, BIOCHEM BIOPH RES CO CLEUTJENS CBJ, 1996, IN PRESS J BIOL CHEM COFFEY DS, 1993, V71, P880, CANCER COOKE PS, 1991, V128, P286, ENDOCRINOLOGY CRAWFORD ED, 1989, V321, P419, NEW ENGL J MED CULIG Z, 1994, V54, P5474, CANCER RES CULIG Z, 1993, V7, P1541, MOL ENDOCRINOL CUNHA GR, 1994, V74, P1030, CANCER CUNHA GR, 1987, V8, P338, ENDOCR REV CUNHA GR, 1981, V14, P1317, J STEROID BIOCHEM DENIS L, 1994, V5, P17, PROSTATE S DERUITER PE, 1995, V110, R1, MOL CELL ENDOCRINOL DEWINTER JAR, 1994, V144, P735, AM J PATHOL DEWINTER JAR, 1991, V39, P927, J HISTOCHEM CYTOCHEM DONJACOUR AA, 1993, V132, P2342, ENDOCRINOLOGY EVANS RM, 1988, V240, P889, SCIENCE FAWELL SE, 1990, V60, P953, CELL GADDIPATI JP, 1994, V54, P2861, CANCER RES GLEAVE M, 1991, V51, P3753, CANCER RES HOBISCH A, 1995, V55, P3068, CANCER RES HUSMANN DA, 1991, V128, P1902, ENDOCRINOLOGY IKONEN T, 1994, V135, P1359, ENDOCRINOLOGY JENSTER G, 1993, V293, P761, BIOCHEM J JENSTER G, 1994, V33, P4064, BIOCHEMISTRY-US JENSTER G, 1995, V269, P7341, J BIOL CHEM JENSTER G, 1991, V5, P1390, MOL ENDOCRINOL KALLIO PJ, 1994, V269, P1514, J BIOL CHEM KASPER S, 1994, V269, P1763, J BIOL CHEM KAZEMIESFARJANI P, 1995, V4, P523, HUM MOL GENET KRAUS WL, 1995, V92, P2314, P NATL ACAD SCI USA LANGLEY E, 1995, V270, P9983, J BIOL CHEM LASPADA AR, 1991, V352, P77, NATURE LIEBERMAN BA, 1993, V7, P515, MOL ENDOCRINOL LOBACCARO JM, 1993, V2, P1799, HUM MOL GENET LUISI BF, 1991, V352, P497, NATURE LUND SD, 1991, V11, P5426, MOL CELL BIOL MCPHAUL MJ, 1993, V76, P17, J CLIN ENDOCR METAB MURTHA P, 1993, V2, P6459, BIOCHEMISTRY-US NEWMARK JR, 1992, V89, P6319, P NATL ACAD SCI USA NORDEEN SK. 1990, V4. P1866, MOL ENDOCRINOL PATTERSON MN, 1994, V22, P3560, NUCLEIC ACIDS RES PERTSCHUK LP, 1995, V73, P302, LAB INVEST PETERZIEL H, 1995, V63, P544, INT J CANCER PINSKY L, 1992, V15, P456, CLIN INVEST MED

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                (ANDROGEN (W) RECEPTOR (W) MUTATION) AND (ANTIANDROGEN)
S1
            8
S2
            4
                RD S1 (unique items)
S (ANDROGEN (W) RECEPTOR) AND MUTATION AND ANTIANDROGEN
          165969 ANDROGEN
         3289846 RECEPTOR
           41932 ANDROGEN (W) RECEPTOR
         1072816 MUTATION
           15574 ANTIANDROGEN
      S3
             293 (ANDROGEN (W) RECEPTOR) AND MUTATION AND ANTIANDROGEN
S S3 AND (DRUG (4W) SELECTION) OR (DRUG (4W) SCREENING)
Processing
Processing
Processing
Processed 10 of 10 files ...
Completed processing all files
             293 S3
        10602543 DRUG
         1002715 SELECTION
            8801 DRUG(4W) SELECTION
        10602543 DRUG
          957656 SCREENING
          103981 DRUG (4W) SCREENING
      S4 103981 S3 AND (DRUG (4W) SELECTION) OR (DRUG (4W) SCREENING)
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S (DRUG (W) SELECTION) AND (ANTIANDROGEN)
Processing
Processed 10 of 10 files ...
Completed processing all files
        10602543 DRUG
         1002715 SELECTION
            3426 DRUG(W) SELECTION
           15574 ANTIANDROGEN
      S5
               2 (DRUG (W) SELECTION) AND (ANTIANDROGEN)
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RD S5
      S6
               2 RD S5
                         (unique items)
TYPE S6/FULL/1-2
  6/9/1
            (Item 1 from file: 5)
DIALOG(R)File
               5:Biosis Previews(R)
(c) 2007 The Thomson Corporation. All rts. reserv.
           BIOSIS NO.: 200200258501
16664990
 Assessment of indicators for hospital drug formulary non-adherence
AUTHOR: Fijn R (Reprint); Lenderink A W; Egberts A C G; Brouwers J R B J;
  De Jong-Van DenBerg L T W
AUTHOR ADDRESS: Medical Sciences and Pharmacy, Department of Social
  Pharmacy and Pharmacoepidemiology, University of Groningen, Antonius
  Deusinglaan 1, 9713 AV, Groningen, Netherlands**Netherlands
JOURNAL: European Journal of Clinical Pharmacology 57 (9): p677-684
November, 2001 2001
MEDIUM: print
ISSN: 0031-6970
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
```

ABSTRACT: Background: Translation of rational drug therapy into practice remains an international problem. Although pharmacotherapeutic treatment quidelines (PTGs) as managerial tools are favoured over hospital drug formularies (HDFs), the latter are still applied in most hospitals. HDF enforcement often leads to time-consuming consultation from the perspective of both pharmacy staff and prescriber. So far, research on HDFs has only been conducted outside Europe. Moreover, this research has only been descriptive. Straightforward indicators qualitatively characterising HDF non-adherence have never been assessed. Methods: A retrospective 1:1 case-control study was conducted across three general teaching hospitals. Non-HDF requests were compared with HDF requests. Data were multivariably analysed, considering patient, prescriber, drug, and HDF characteristics as possible indicators for non-adherence. Results: HDF adherence was almost universal across characteristics. Non-adherence was characterised by newly marketed drugs, drugs that were part of patients' pre-admission drug therapy, drugs with many fellow drugs within the drug group on the market, and drugs originating from a drug group for which the HDF was highly restrictive. Contrary to common perception, non-adherence was independent of medical specialty, therapeutic area, and patient characteristics. Conclusion: This research provides an epidemiological framework for hospitals (drug and therapeutics committees) for evaluating pharmacy data on HDF non-adherence. It can be used for educational tailor-made feedback to prescribers and for drug selection when the inclusion of newly marketed

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approach involving secondary and primary health care to establish
  continuity in seamless care of drug therapy.
REGISTRY NUMBERS: 12794-10-4: benzodiazepines; 14797-55-8: nitrates;
    148-79-8Q: statins; 79902-63-9Q: statins; 63-74-1: sulphonamides
DESCRIPTORS:
  MAJOR CONCEPTS: Hospital Administration -- Allied Medical Sciences;
    Pharmacology
  BIOSYSTEMATIC NAMES: Hominidae--Primates, Mammalia, Vertebrata, Chordata,
  ORGANISMS: human (Hominidae) -- female, male, patient
  COMMON TAXONOMIC TERMS: Animals; Chordates; Humans; Mammals; Primates;
    Vertebrates
                              alpha-adrenoreceptor antagonists--adrenergic
  CHEMICALS & BIOCHEMICALS:
    antagonist-drug, alpha-adrenergic antagonist-drug, autonomic-drug;
    antiandrogens--antiandrogen-drug; benzodiazepines--anxiolytic-drug,
    sedative/hypnotic-drug; beta-blockers--adrenergic antagonist-drug,
    autonomic-drug, beta-adrenergic antagonist-drug; biphosphonates;
    calcium channel blockers--calcium channel blocker-drug,
    cardiovascular-drug; corticosteroids--antiinflammatory-drug,
    immunologic-drug; nitrates; proton-pump inhibitors; statins--HMG COA
    reductase inhibitor-drug, cardiovascular-drug, enzyme inhibitor-drug;
    sulphonamides -- antidiabetic - drug, diuretic - drug, renal - acting - drug
  METHODS & EQUIPMENT: rational drug therapy--therapeutic method
                         hospital drug formulary non-adherence;
  MISCELLANEOUS TERMS:
    pharmacoepidemiology; pharmacotherapeutic treatment guidelines
CONCEPT CODES:
  10060 Biochemistry studies - General
  10067 Biochemistry studies - Sterols and steroids
  12512 Pathology - Therapy
  22002 Pharmacology - General
  22005 Pharmacology - Clinical pharmacology
  22010 Pharmacology - Cardiovascular system
  22012 Pharmacology - Connective tissue, bone and collagen-acting drugs
  22016 Pharmacology - Endocrine
  22018 Pharmacology - Immunological processes and allergy
  22024 Pharmacology - Neuropharmacology
  22026 Pharmacology - Psychopharmacology
  22032 Pharmacology - Urinary system
  37010 Public health - Public health administration and statistics
BIOSYSTEMATIC CODES:
  86215 Hominidae
  6/9/2
            (Item 2 from file: 5)
DIALOG(R) File
                5:Biosis Previews(R)
(c) 2007 The Thomson Corporation. All rts. reserv.
           BIOSIS NO.: 200100376552
16204713
 Combined androgen blockade in prostate cancer: Meta-analyses and associated
 issues
AUTHOR: Klotz L (Reprint)
AUTHOR ADDRESS: Sunnybrook and Women's College Health Sciences Centre, 2075
  Bayview Avenue, North York, No. MG 408, Toronto, Ontario, M4N 3M5, Canada
  **Canada
JOURNAL: BJU International 87 (9): p806-813 June, 2001 2001
MEDIUM: print
ISSN: 1464-4096
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drugs is considered or HDF restrictiveness for certain drug groups is reconsidered. Moreover, it demonstrates the importance of a regional

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DOCUMENT TYPE: Article
RECORD TYPE: Citation
LANGUAGE: English
DESCRIPTORS:
  MAJOR CONCEPTS: Oncology -- Human Medicine, Medical Sciences; Urology --
    Human Medicine, Medical Sciences; Pharmacology
  BIOSYSTEMATIC NAMES: Hominidae--Primates, Mammalia, Vertebrata, Chordata,
    Animalia
  ORGANISMS: human (Hominidae) -- male, patient
  COMMON TAXONOMIC TERMS: Animals; Chordates; Humans; Mammals; Primates;
  DISEASES: prostate cancer--neoplastic disease, reproductive system
    disease/male, urologic disease, treatment
  MESH TERMS: Prostatic Neoplasms (MeSH)
  CHEMICALS & BIOCHEMICALS:
                              antiandrogen
  METHODS & EQUIPMENT: combined androgen blockade--drug selection,
    efficacy, patient selection, therapeutic method
  MISCELLANEOUS TERMS:
                         Meta-analysis
CONCEPT CODES:
  12512 Pathology - Therapy
  15506 Urinary system - Pathology ·
  16506 Reproductive system - Pathology
  22002 Pharmacology - General
  22005 Pharmacology - Clinical pharmacology
  22016 Pharmacology - Endocrine
  24004 Neoplasms - Pathology, clinical aspects and systemic effects
  24008 Neoplasms - Therapeutic agents and therapy
BIOSYSTEMATIC CODES:
  86215 Hominidae
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Set	Items	Description
S1	8	(ANDROGEN (W) RECEPTOR (W) MUTATION) AND (ANTIANDROGEN)
S2	4	RD S1 (unique items)
S3	293	(ANDROGEN (W) RECEPTOR) AND MUTATION AND ANTIANDROGEN
S4	103981	S3 AND (DRUG (4W) SELECTION) OR (DRUG (4W) SCREENING)
S5	2	(DRUG (W) SELECTION) AND (ANTIANDROGEN)
S6	2	RD S5 (unique items)
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